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A COMPARISON BETWEEN THE RESULTS OF BLOOD  
CULTURES TAKEN DURING LIFE AND  
AFTER DEATH.\*

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So much has been done in recent years in the bacteriology of the blood and internal organs that we are all cognizant of the value of this method of diagnosis. In many cases the results of blood cultures are the only positive proofs of the nature of the infection, particularly in the large group of septicemic cases, where in the absence of some of the signs and reactions common to the better known infections such as the pulmonary signs, the sputum of pneumonia, the reaction of agglutination, and the skin pictures of many febrile diseases, the clinician is often much perplexed concerning the nature of the infection or intoxication at hand. Frequently at the present day the diagnosis can be made during life, and such a diagnosis made early may, with the future development of curative anti-sera, assume an importance equal to that of the recognition of diphtheria.

Much oftener, however, owing to a variety of reasons, these diagnoses are made postmortem, and the final decision rests with the pathologist or bacteriologist, who, however, has the clearness of his horizon to a greater or less degree clouded by the following facts: First, the occurrence of an agonal or pre-agonal migration of bacteria from the intestinal tract or lungs into the general circulation; secondly, the multiplication of organisms in the vessels and organs after death either already there, antemortem, or by invasion postmortem.

With regard to the latter it may be said that the wide-spread area of occupation in some instances suggests the likelihood of a deposition of bacteria in these parts before death, whence they

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wander freely postmortem, not by the arteries which are empty, but by the venous channels in which the blood remains fluid for some hours, or if coagulated, a sufficiency of serum still remains to facilitate progression.

Another factor to be considered in dealing with the presence of bacteria in the blood is the action of the bacteriolytic complement existing in the plasma. Longcope<sup>1</sup> believed that the presence or absence of terminal infections depended largely upon the amount of complement in the blood, a conclusion not wholly acceptable, since Weaver and Ruediger,<sup>2</sup> Rosenow,<sup>3</sup> Behring<sup>4</sup> and others have shown that upon streptococcus and pneumococcus normal sera exert no bacteriolytic action whatever. Nevertheless, as Longcope's experiments show, there does exist a certain definite antagonism against some bacteria, e. g., *B. typhosus* and *B. coli*, so that the activity of the complement content of the blood cannot be altogether discounted.

It seemed to us that a comparison of the bacteriological findings in the blood before and after death could not fail to be more than usually instructive, particularly since these results, if showing any similarity would have a doubly acting significance, the postmortem reading confirming the provisional antemortem bacteriological diagnosis. And the antemortem blood condition when in agreement with the postmortem would prove the value of this method of investigation and show to what extent contaminations, "agonal infections," and postmortem bacterial proliferation are to be considered in autopsy bacteriology.

Thanks to the courtesies of Professors Osler and Welch, we were enabled to make both ante- and postmortem bacteriological examinations of the blood in many cases, and to obtain positive results in a series of most interesting conditions. At autopsy the bacteriological investigations were conducted by one of us, while the blood cultures during life were taken by the other, using in both instances the utmost precautions necessary to reliable results. The technique of this operation during life, one which in innumerable cases has given us most satisfactory results, is briefly

<sup>1</sup> *Univ. Pennsylv. Med. Bull.*, 1902, 15, p. 331.

<sup>2</sup> *Medicine*, 1903, 9, p. 515.

<sup>3</sup> *Jour. Infect. Dis.*, 1904, 1, p. 280.

<sup>4</sup> Quoted by Rosenow.

as follows, the arm constricted tightly in the upper third, is scrubbed with green soap at the elbow fold, then alcohol is applied, either poured over, or rubbed over (the former by preference), succeeded by an application of ether in a similar fashion, followed by bichlorid of mercury 1:10,000–20,000, and lastly more ether or alcohol to dry off the skin. A large barrelled syringe (10 to 30 c.c.) was used and the needle inserted into one of the now prominent veins, several syringefuls of blood were frequently taken and either put into liquid media (broth, or milk and broth) or plated out in agar. These two methods of culture were employed, the first to diminish any bactericidal action by sufficiently diluting the blood, the second to avoid spoiling results by contamination, and to give an idea of the number of organisms present in the blood. At autopsy the cultures were preferably made from the heart's blood, the pericardium being burned with a red hot blade and opened with a lance, then the auricular heart muscle being similarly treated, a large platinum loop needle, or at times a small pipette, was introduced into the auricle and cultures made in agar plates, coverslips of the blood being usually taken at the same time, and cultures made from other organs.

The objections to the reliability of blood cultures, namely the possibility of taking contaminating organisms into consideration as infecting agents, requires a few words. When once a good technique had been established it was soon found that cultural results during life were in an overwhelmingly large per cent of cases quite sterile. In a few instances, not regularly, as one might imagine, knowing the difficulty experienced in sterilizing the skin, staphylococcus pyogenes albus in limited numbers, one or two colonies per plate, would appear in spite of all precautions. In direct proportion to lack of care in sterilizing apparatus, the ever present hay bacillus might be expected to overgrow the plates and tubes while other members of the contaminating horde might be met with. In a series of blood cultures embracing perhaps several hundreds of cases these two bacteria were practically the only contaminating organisms coming under our observation. It may be well to remark that we have never in our experience found unexpectedly or as contaminants in our cultures such prevalent

organisms as streptococcus pyogenes, *B. coli*, or staphylococcus pyogenes aureus, the plate method of culture having always to our mind been of great assistance in helping to rule out contaminations. Positive results give as a general rule several or many colonies per c.c. of blood in a plate; contaminating organisms, unless gross carelessness has been displayed, rarely number more than one or two colonies in a plate; in liquid media, of course, a contaminating microorganism may generally overgrow an infecting agent from the blood.

The ease with which positive results are now obtained in some of our well known clinically recognizable diseases such as pneumonia and typhoid fever, overrules most objections hitherto raised against the reliability of the procedure, and it is part of our task to show to what extent we may rely upon cultures to prove the existence of other less easily determinable infectious processes.

Many objections are urged against accepting unreservedly the results of postmortem bacteriology and many of these objections cannot be opposed. In from four to six hours after death some of the common organisms of the intestine are to be found in the neighboring organs unless the body has been carefully and immediately put in the refrigerator; this militates at once against many so-called colon-infections and infections supposedly due to similar and allied organisms. The presence of a single prevailing organism in exudates and inflammatory lesions may usually be accepted as evidence that the particular organism is the infecting agent, but infection of postmortem fluid transudates, in any of the serous cavities, is common and may occur early; the absence, however, of inflammatory reaction, in such cases, should prevent false interpretations of bacteriological results. We also recognize in routine work certain organisms as being more likely to be contaminating than infecting agents.

In cases of general infections, the subject with which we have to do, we are dealing with a fluid medium, the blood, which furnishes an avenue of connection between all parts of the body, and hence is open to all manner of infections, the sources of which, in many cases are apparent (local lesions) in many others are

obscure, as typhoid fever, pneumonia, rheumatism, epidemic cerebro-spinal meningitis (granting consideration to the general infection idea of these diseases). Postmortem invasion and infection of the blood from many sources is possible; postmortem proliferation of a few wandering organisms may give the bacteriological findings the appearance of a severe general infection; a postmortem contamination may obscure all bacteriological results. These are the general suppositions, it remains to be seen to what extent they are absolute, to what extent they are to be considered, practically, in other words, what credence may be given to reports of infections based on autopsy findings. As we have already stated, a correspondence between the blood cultures results before death and the postmortem cultural findings would cover all these points, and prove the value of the method of diagnosis.

Our table gives in detail the histories of the cases and the combined bacteriological reports.

Of fourteen cases considered there are seven in which the antemortem and postmortem results are absolutely the same; two in which the same organisms were present before and after death, but with the addition of another infecting or contaminating microbe in the postmortem cultures; five in which the antemortem results are not confirmed by the postmortem findings, or which show radical differences in cultural results.

In regard to the first group of cases, the seven showing absolute agreement in the antemortem and postmortem results, it need only be said that they are conclusive evidence of the value of blood culture and show that postmortem results may be accepted in a large per cent of cases. Closer inspection of the two next mentioned cases, Nos. 7 and 9, show that the postmortem difference is clearly due to an additional infection.

In No. 7 the deposition of streptococci and staphylococcus pyogenes aureus in the kidney and urine must be considered as happening in the so-called agonal period, their source from a local infection in the pleural exudate is apparent; cultures from the blood eight hours antemortem showed no sign of their presence, merely the heavy typhoid infection.

TABLE 1.  
SHOWING CLINICAL, PATHOLOGICAL, AND BACTERIOLOGICAL DATA OF CASES.

Patient	Nature and History of Infections	Time of Cultures: Media Used	Antemortem Cultural Results	Anatomical Diagnosis and Postmortem Cultural Results
1. Jacob B. No. 24,628.	Cerebro-spinal meningitis. Fourth day of disease, onset with headache, vomiting and chill; retraction of neck, early; marked hemorrhagic rash on elbows and knees; nephritis (hematuria slight). On 5th day, general redness and swelling of joints; mottling of body surface; pneumonia right lower lobe. Temperature 101° on admission, rising to 105° at death. Unconscious and delirious.	Culture 20 hours A. M. a) Lumbar puncture: Loeffler's serum, glycerine agar, agar plates. b) Culture from knee joint: serine agar plate, blood on surface; two agar plates; three plain blood undiluted.	a) 20 c.c. of turbid fluid: intracellular diplococci; growth of few typical colonies; b) 40 c.c. of stringy pus; numerous intracellular diplococci, growth of a few typical colonies; c) few typical colonies; growth in undiluted blood. Cultures and protocols indicate that from spinal fluid, knee-joint and blood, the meningococcus was isolated.	Meningococcus from meninges of cord and brain. Micrococcus lanceolatus from lung and blood of coronary artery.
2. A. S. 37 yrs.* Sept. 14, 1898. Oct. 3, 1898.	Rheumatism in 1889. Fever from July, 1898, to August; coming to hospital as supposed relapse in typhoid fever. Aortic valve disease present with fever; diagnosed malignant endocarditis and septicemia.	Culture in 9 days and 3 days A. M. from blood.	Both positive; micrococcus zymogenes.	Endocarditis of aortic valve. Septic infarcts in spleen and kidney. Micrococcus zymogenes in lung, spleen, kidney, gall bladder, aortic and mitral valves, and heart's blood.
3. Daniel S. 63 yrs. Carmar. No. 24,513. Dec. 17, 1897. Dec. 20, 1897.	Pneumonia; onset 6 days previous with chill, pain in side, cough, consolidation in right side. Temperature 104° on admission, 101° at death. Gradual failure. Friction rub in right upper back. Heart irregular, no murmurs or pericardial friction. Leucocytes: on 17th, 32,000; on 20th, 20,000. Urine: albumen trace, few casts, numerous red blood cells, pus.	Culture on the 17th day A. M. a) agar; b) in Marmoreck's serum.	3 to 4 c.c. of blood. a) Numerous pin-point colonies: by protocols micrococcus lanceolatus. b) Contaminated. Mouse inoculated. ½ c.c. bouillon, culture fatal in 8 hours, diplococci in blood; micrococcus lanceolatus.	Micrococcus lanceolatus in heart's blood, lung, pleural exudate, and spleen.

\* Reported by MACCALLUM AND HASTINGS, *Jour. Exp. Med.*, 1899, 4, 521.

TABLE 1—Continued.

Patient	Nature and History of Infections	Time of Cultures: Media Used	Antemortem Cultural Results	Anatomical Diagnosis and Postmortem Cultural Results
4. R.	Typhoid. History indefinite. Severe case, delirium, probably in 3d week. High fever. Widal positive. Death from intoxication, July 10, 1897.	Culture 4 to 5 hours A. M.	Abundant growth of typhoid bacilli.	Typhoid lesions, typhoid bacilli in heart's blood, pericardial fluid, spleen, and mesenteric glands.
5. J. B.	Typhoid, severe case, 3d week: delirium, typhoid state, husky voice. Death from intoxication, Nov. 20, 1897.	Cultures on 8, 6, 4, and 1 days A. M.	8, 6, 4 days negative; 1 day colon bacilli.	Typhoid lesions and ulcers in larynx. Typhoid bacilli in liver, spleen, retroperitoneal and mesenteric glands, gall bladder, and urinary bladder. Streptococci in ulcers of larynx, esophagus, and heart's blood.
6. Kate W. Colored. 84 yrs. Servant. No. 25,221 Jan. 9, 1899. Feb. 1, 1899.	Arterio-sclerosis, chronic parenchymatous nephritis of several years, edema of feet and ankles, acute attack of headaches and weakness two weeks previous to admission, ascites and edema of legs. Systolic murmur at apex. No frictions. Temperature, irregular; 101.8° highest. Jan. 28th, development of pneumonia, diarrhea, vomiting. Delirium. Death on Feb. 1st. Leucocytes, 6,000. Temperature, Jan. 29th, 100°. " " 30th, " " 31st, } Normal. " Feb. 1st, } Urine: albumen, hyaline and granular casts.	Culture Feb. 1st. a) Blood on agar plates. b) Blood in broth, 10 c.c.	a) Negative. b) Streptococcus pyogenes, 10 to 12 elements in chain. 2 c.c. of broth culture not fatal to guinea pigs.	Bacillus coli in kidney.



TABLE 1—Continued.

Patient	Nature and History of Infections	Time of Cultures: Media Used	Antemortem Cultural Results	Anatomical Diagnosis and Postmortem Cultural Results
7. Ryan. Mar. 25, 1899.	Continued case of typhoid; intermittent chills for 10 weeks. Thrombosis of right femoral vein, slight jaundice, developing coma. Friction rub at base of left lung. Urine: albumen, trace; bacilli present.	a) Cultures from urine repeatedly; b) from blood 6 to 8 days A. M. Bacilli in fresh blood.	a) Positive. b) Positive; bacillus typhosus.	Bacillus typhosus in blood, liver, bile, spleen, kidney, peritoneal fluid, urine and area of pleurisy. Streptococcus aureus and Staphylococcus pyogenes aureus in kidney, pelvis, pleuritic adhesion, and in urine.
8. Jones. Oct. 19, 1898.	Arterio-sclerosis: endocarditis. Irregular temperature. Edema, dyspnoea.	Cultures 3 days A. M.	Positive; staphylococcus-pyogenes aureus.	Bacilli in heart's blood, urine, and peritoneal cavity. Staphylococcus aureus and albus in mitral valve vegetation.
9. P. O. Dec. 1, 1898. Feb. 1, 1899.	Lymphatic leukemia. Weakness and glandular swellings since 1896. Two previous admissions; improved and able to work. September, 1898, dyspnea and edema of legs, palpitation, diarrhea, cough: continuous arsenic treatment. Blood: R. B. C., 2,712,000; W. B. C., 44,000; hemoglobin, 40%. Leucocytes: small mononuclear, 89.5%; polymorphonuclear, 8.0%; eosinophiles, 2.0%; transitionals, 0.5%. Steady increase of leucocytes to 139,000. Pulse failed steadily; irregular temperature present on admission, more marked on Dec. 12, 1898—102.5°. Dyspnea, glandular swellings, cough all increased. Many infected papules on legs and hands. Died with no increase of symptoms on Feb. 1, 1899.	Jan. 18, 1899, 3 days A. M. Jan. 31, 1899, 1 day A. M.	Negative. Agar plates from blood, positive. Streptococci numerous.	Streptococcus in heart's blood, spleen, peritoneal cavity, inguinal glands, and edematous tissues in neighborhood. Mic. lanceolatus in lung.

TABLE 1—Continued.

Patient	Nature and History of Infections	Time of Cultures: Media Used	Antemortem Cultural Results	Anatomical Diagnosis and Postmortem Cultural Results
10. S. W. P. June 21, 1897. June 24, 1897.	Asthma: edema of legs. Acute rheumatism 30 years ago. Moderate drinker. Had jaundice 18 months ago; gastro-intestinal disturbances, 13 months ago; hematemesis, 10 days ago; edema, delirium, and jaundice marked; chest clear; some ascites, liver enlarged, spleen palpable. Albuminuria. Leucocytes, 20,000. Temperature, 101°-104°; 107° at death on June 24.	June 22, 2 days A. M. June 23, 1 day A. M.	Positive } Streptococci. Positive }	Hypertrophic cirrhosis. Streptococci in heart's blood, lung, bile, kidney, spleen.
11. J. A. 35 yrs. Colored. May 18, 1897. May 28, 1897.	Aortic insufficiency, edema, dilated heart, ascites, ulcer on leg. May 19, sudden rise of temperature to 104°, cough. Temperature remained elevated, reaching 106.5° on May 25. Died on May 28. No local signs to account for temperature. Urine albuminous, and a few blood casts.	Blood culture A. M.	Positive; streptococci.	Aortic insufficiency, endocarditis arterio-sclerosis. Streptococci from aortic valves, heart's blood, infarcts of spleen, and kidney.
12. F. 34 yrs.* Apr. 28, 1895. May 16, 1895.	Rheumatism for 3 months, weakness and dyspnea, severe chills, anemia, sweatings; physical signs of mitral stenosis. Leucocytosis. Urine: albumin and casts. Diagnosis: ulcerative endocarditis. Death May 16, 1895.	Cultures from blood, May 7, May 12, 1895.	Positive; mic. gonorrhoeae.	Ulcerative endocarditis of mitral valve; typical biscuit-shaped cocci decolorizable by Gram; cocci not able to be cultivated on media at hand.

\* Reported by THAYER AND BLUMER, *Johns Hopk. Hosp. Bull.*, 1896, 7, 57.

TABLE 1—Continued.

Patient	Nature and History of Infections	Time of Cultures: Media Used	Antemortem Cultural Results	Anatomical Diagnosis and Postmortem Cultural Results
13. J. K. 19 yrs.* Feb. 5, 1896. Feb. 25, 1896.	Fever, weakness; gonorrhea 6 months ago; shaking chills Nov. 25, 1895; fever sweats; progressive anemia and weakness; liver and spleen enlarged. Systolic murmur at base. Left knee swollen; urethral discharge, gonococci in same. Blood: R. B. C., 2,290,000; W. B. C., 9000. Urine: albumen and pus. Left hospital Feb. 14, 1896; re-entered March 9, 1896, worse; urethral discharge less; friction over heart; petechiae on skin. Death March 25, 1896.	Mar. 22 Mar. 24 Mar. 25.	Negative. Positive } Positive } Mic. gonorrhoeae	Tricuspidocarditis, hemorrhagic glomerulonephritis; acute sero-fibrinous pleurisy; pericarditis. Gonococci from pericardium, tricuspid valves, heart's blood.
14. Feb. 1, 1896.	History unknown. Pericarditis, pneumonia. Death Feb. 1, 1896.	Blood cultures a few hours antemortem.	Positive; streptococci.	A diplococcus from pericardial exudate and pneumonic area: micrococcus lanceolatus.

\* Reported by THAYER AND LAZEAR, *Jour. Exp. Med.*, 1899, 4, 81.

TABLE 2.  
SUMMARY OF CASES IN WHICH ANTEMORTEM AND POSTMORTEM CULTURES DIFFER.

Antemortem	Time of Culture	Postmortem
1. Meninges, knee joint, blood. Meningococcus.	20 hours A. M.	Meningococcus from cord, brain and meninges. Pneumococcus in blood and lungs.
5. Typhoid severe. Blood B. coli. No agglutination with B. coli.	4 cultures; 3 neg., 1 pos., 24 hours A. M.	B. typhosus in liver, spleen, retro-peritoneal glands, mesenteric glands and gall-bladder. Streptococci in ulcers of larynx, oesophagus and heart's blood.
6. Arterio-sclerosis: temperature; pneumonia. Death afebrile. An aerobic streptococcus.	Few hours A. M.	Bacillus coli in kidney. Blood negative.
7. Typhoid fever. B. typhosus in blood, and seen in fresh blood: B. typhosus in urine.	6 to 8 hours A. M.	B. typhosus in blood, liver, bile, spleen, kidney, peritoneal cavity, urine and pleura; streptococcus and staphylococcus aureus in kidney-pelvis, pleura and urine.
8. Arterio-sclerosis; endocarditis; Staphyl. aureus.	3 days A. M.	Bac. in blood and peritoneal cav., Staph. aureus and albus on mitral valve.
9. Lymphatic anemia, chronic. Irregular temperature; bronchopneumonia; streptococci in blood.	24 hours A. M.	Streptococcus in blood, spleen, peritoneal cavity, inguinal glands, and edematous tissues in neighborhood; mic. lanceolatus in lung.
14. Pericarditis; pneumonia. Blood cultures, streptococcus.	A few hours A. M.	Micrococcus lanceolatus from pericardial exudate, and pneumonic area in lung.

In No. 9 a suspected bronchopneumonia as evidenced by temperature and dyspnea probably allowed, and explains, the presence of the pneumococci in the blood and tissues, they not being present 24 hours antemortem.

In the last group of cases, five show considerable lack of correspondence between the ante- and postmortem cultures, and are more difficult of reconciliation.

No. 1, a case of epidemic cerebrospinal meningitis, the first reported instance of general infection with the meningococcus, the development of pneumonia explains the presence of the pneumococci in the blood postmortem. The non-development of meningococcus in the postmortem blood cultures may have been due to the feeble vitality of the organism, rendering cultivation difficult, or to their scarcity in or disappearance from the blood.

Case 5, a severe typhoid; four blood cultures failed to show the presence of the typhoid bacillus, in spite of the apparent

heavy infection found postmortem. The isolation of *B. coli* in this case 24 hours before death under such conditions is interesting and may be taken as an instance of a transitory bacteremia with its point of origin in some portion of the ulcerated intestinal tract; and since there was no serum reaction present in the blood for this bacillus, we must recognize that in this and similar cases infection of the blood stream may take place in the death agony, beclouding actual conditions and giving rise to varied postmortem results. Furthermore, a negative result, as is well known, may be met with several times in succession in this disease, so that the discrepancy is more seeming than real.

In Case 6, the lack of postmortem confirmation may have been due, on the one hand, to the disappearance of the organism from the circulation; on the other, to difficulty in cultivation or in not having taken a sufficiency of blood. The presence of *B. coli* may be held in the light of a postmortem migration.

The divergence of results in Case 8 meets with a possible explanation upon the grounds of a frank agonal or postmortem invasion; for the unidentified bacillus could scarcely have escaped recognition in the antemortem culture had it been present, considering its apparent widespread distribution. *Staphylococcus aureus* appearing in the mitral valve and not in the blood, might be accounted for by its being suppressed by the swarming in of the strange bacillus into the circulation or being crowded out in the plate. The presence of *staphylococcus albus* may possibly be due to an error in diagnosis, knowing as we do that some of the colonies amongst pigment producing bacteria at times fail to show pigment and their descendants for some generations may remain colorless.

The lack of harmony in Case 14 is undoubtedly caused by the development of pneumonia resulting in an admixture of organisms in the blood stream, with consequent difficulty of differentiation of the two types of colonies on the plate, the pneumonic infection being largely in the nature of an agonal one.

It will be seen, therefore, in such groups of divergent case results that no serious objections can be raised against the value of blood cultures during life. Failures or lack of harmony with

postmortem results may be caused, in the first place, by the non-appearance of organisms in the circulation in spite of the very definite clinical picture of an infection, due to causes at the present time imperfectly understood; in the second place, as was previously pointed out, to agonal or postmortem invasion by such organisms of the intestinal group as *B. coli* and its close relatives. In our experience infection during life with *B. coli* was a rare occurrence, and may be looked for where the gates are very widely open, as in conditions of extensive ulceration of the intestinal tract, or where there is lessened bactericidal action on the part of the blood toward such relatively feeble organisms as the members of the colon group.

Illustrative of a variety of conditions influencing the bacteriological results postmortem, we present a series of cases taken at random and shown in Table 3.

Without going into an exhaustive analysis of the cases, there are certain conditions worthy of remark. There is observable in Cases 1, 4, 5, and 9 a very definite polybacterial infection, due to doubtless to several factors; e. g., postmortem invasion, or multiplication of bacteria already there (5 and 9), to secondary local infection (1 and 4), or to the length of time existing between the death of the individual and the taking of cultures.

For unknown reasons, Cases 2, 6, and 8, all with surgical aspects, curiously enough do not supply us with as complex a picture as might be expected.

In cases illustrative of specific infective diseases, as typhoid fever and pneumonia—Cases 1, 4, 7, and 8—the bacteriological findings confirm the anatomical diagnosis by the recovery of the specific microbes from the tissues. The failure to isolate *Mic. lanceolatus* where frank lobar pneumonia existed, in Cases 4 and 5, was caused, in all likelihood, by the accompanying polybacterial infection either totally suppressing growth or rendering it unobservable on the agar plates.

Any activity of the bactericidal power of the blood exhibited in these cases seems to be present in some degree in Cases 3 and 10, in the latter as long as 13½ hours after death.

It is evident from a perusal of the above table that with few

TABLE 3.

Num- ber	Anatomic Diagnosis	Time after Death	Bacteriological Results
1....	Typhoid fever. Bronchopneumonia.	5 hrs.	B. typhosus: spleen, gall-bladder, mesenteric glands, and bone-mar- row. Streptococcus Staphylococcus pyogenes } Lung. aureus
2....	Perforation of duodenal ulcer. General peritonitis.	5 hrs.	B. coli: heart's blood, spleen, gall- bladder, and peritoneal cavity. Streptococcus: peritoneal cavity.
3....	Perforation of duodenal ulcer. General peritonitis. Bronchopneumonia.	2 hrs.	Sterile: heart's blood. Mic. lanceolatus: lung.
4....	Typhoid fever. Lobar pneumonia.	31 hrs.	B. typhosus: gall-bladder and spleen. B. coli Streptococcus Staphylococcus pyogenes } Lung. aureus
5....	Lobar pneumonia. Bronchopneumonia. Abscess of parotid gland. Acute endocarditis.	12½ hrs.	Staphylococcus pyogenes } Lung. albus B. lactis aërogenes Bacillus (unidentified): lung, heart's blood, and parotid abscess.
6....	General peritonitis following colostomy.	12 hrs.	Streptococcus: spleen, liver, and kidney.
7....	Lobar pneumonia. Pericarditis.	8 hrs.	Mic. lanceolatus: heart's blood and lung. Sterile: spleen, and bronchial glands.
8....	Typhoid fever. Perforation.	20 hrs.	B. typhosus: spleen, liver, and gall- bladder. B. coli: liver.
9....	Hemorrhagic infarct of lung. Thrombosis of branch of pulmo- nary artery going to lower lobe. Acute bronchopneumonia.	13½ hrs.	Streptococcus: heart's blood and thrombus. B. pseudo-diphthericus: infarct. Staphylococcus pyogenes } Throm- aureus bus. B. coli
10....	Chronic endocarditis.	13½ hrs.	Sterile: heart's blood.

exceptions uncontrolled postmortem findings must always be of more or less doubtful value, particularly if taken several hours after death and not associated with marked clinical symptoms. We must recognize, too, that almost all, if not all, the common bacteria of the intestines—particularly *B. coli*, *B. lactis aërogenes*, *Streptococcus pyogenes*, and *B. pseudodiphthericus*—may be met with in the circulatory channels and organs, probably as contaminating postmortem invaders, although one must admit that the mode of their entrance is not at all times clear. And it is here especially worthy of mention that the mere presence of these organisms in the blood should never be accepted as indisputable evidence of general infection, unless there exists confirmatory antemortem cultural findings, or definite clinical facts, such as

local lesions or the acknowledged signs of specific infectious diseases.

The presence of a variety of organisms on a plate at one time is always suggestive of contamination, unless there exists evidence of two distinct infective processes, as typhoid fever and pneumonia; but even such conditions as perforative peritonitis may show surprisingly paradoxical results on investigation of the inoculated media.

The presence or absence of the bactericidal power of the blood is, as Longcope has pointed out, probably responsible to a considerable degree for the great variation seen in a lengthy series of cases.

#### CONCLUSIONS.

As a result of our study of correlated ante- and postmortem cultures we offer the following conclusions:

1. That within certain time limits postmortem bacteriological methods afford trustworthy means of determining or confirming the presence of many of the well-known infectious processes.

2. That the more often a marked antemortem infection is present, the greater probability there is of finding an uncomplicated postmortem bacteriological result, provided the investigation is carried on within a reasonable time after death, say 12 hours.

3. That a bacteremia due to the common organisms of the intestinal tract and the so-called "agonal invasions" of the blood stream do occur, yet they should not be assumed to be present with any great degree of frequency.

4. That the rôle played by the bacteriolytic complements is probably considerable, although not entirely explaining in some cases the negative results during life with positive results after death, or *vice versa*.

5. That in view of the constant number of clinically unrecognized general infections in our hospitals, and the future possibilities of serum treatment, bacteriological investigation of the blood stream both before and after death should be more generally encouraged and practiced.